

2. Hiramatsu K, Aritaka N, Hanaki H *et al.* Dissemination in Japanese hospitals of strains of *Staphylococcus aureus* heterogeneously resistant to vancomycin. *Lancet* 1997; **350**: 1670–1673.
3. Tenover FC, Biddle JW, Lancaster MV. Increasing resistance to vancomycin and other glycopeptides in *Staphylococcus aureus*. *Emerg Infect Dis* 2001; **7**: 327–332.
4. Ploy MC, Grelaud C, Martin C, De Lumley L, Denis F. First clinical isolate of vancomycin-intermediate *Staphylococcus aureus* in a French hospital. *Lancet* 1998; **351**: 1212.
5. Johnson AP. Intermediate vancomycin resistance in *Staphylococcus aureus*: a major threat or a minor inconvenience. *J Antimicrob Chemother* 1998; **42**: 289–291.
6. European Antimicrobial Resistance Surveillance System. *Technical guide for the detection of VISA/VRSA*. Bilthoven: National Institute for Public Health and the Environment (RIVM), 2004. <http://www.earss.rivm.nl>.
7. Walsh TR, Bolmström A, Qwärnström A *et al.* Evaluation of current methods for detection of staphylococci with reduced susceptibility to glycopeptides. *J Clin Microbiol* 2001; **39**: 2439–2444.
8. Chesneau O, Morvan A, Solh NE. Retrospective screening for heterogeneous vancomycin resistance in diverse *Staphylococcus aureus* clones disseminated in French hospitals. *J Antimicrob Chemother* 2000; **45**: 887–890.
9. MacKenzie FM, Greig P, Morrison D, Edwards G, Gould IM. Identification and characterization of teicoplanin-intermediate *Staphylococcus aureus* blood culture isolates in NE Scotland. *J Antimicrob Chemother* 2002; **50**: 689–697.
10. Kumari DN, Keer V, Hawkey PM *et al.* Comparison and application of ribosome spacer DNA amplicon polymorphisms and pulsed-field gel electrophoresis for differentiation of methicillin-resistant *Staphylococcus aureus* strains. *J Clin Microbiol* 1997; **35**: 881–885.
11. Marchese A, Balistreri G, Tonoli E, Debbia EA, Schito GC. Heterogeneous vancomycin resistance in methicillin-resistant *Staphylococcus aureus* strains isolated in a large Italian hospital. *J Clin Microbiol* 2000; **38**: 866–869.
12. Vandenesch F, Naimi T, Enright MC *et al.* Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton–Valentine leukocidin genes: worldwide emergence. *Emerg Infect Dis* 2003; **9**: 978–984.
13. Walsh TR, Howe RA. The prevalence and mechanisms of vancomycin resistance in *Staphylococcus aureus*. *Ann Rev Microbiol* 2002; **56**: 657–675.
14. Ariza J, Pujol M, Cabo J *et al.* Vancomycin in surgical infections due to methicillin-resistant *Staphylococcus aureus* with heterogeneous resistance to vancomycin. *Lancet* 1999; **353**: 1587–1588.
15. Reverdy ME, Jarraud S, Bobin-Dubreux S *et al.* Incidence of *Staphylococcus aureus* with reduced susceptibility to glycopeptides in two French hospitals. *Clin Microbiol Infect* 2001; **7**: 267–272.
16. Hageman JC, Pegues DA, Jepson C *et al.* Vancomycin-intermediate *Staphylococcus aureus* in a home health-care patient. *Emerg Infect Dis* 2001; **7**: 1023–1025.

RESEARCH NOTE

Microbiology of sinusitis and the predictive value of throat culture for the aetiology of sinusitis

A. Ilki¹, N. Ulger¹, S. Inanir², E. Ozer²,
C. Arikan³, M. Bakır⁴ and G. Soyletir¹

¹Department of Microbiology, ²Department of Otolaryngology, ³Department of Paediatrics and ⁴Division of Paediatric Infectious Disease, Marmara University, Istanbul, Turkey

ABSTRACT

A prospective study of throat cultures and maxillary sinus aspirates from children with chronic sinusitis ($n = 21$), acute sinusitis ($n = 28$) or a clinical diagnosis of chronic adenoiditis ($n = 41$) was performed. Seventy-two bacterial pathogens were isolated from sinus aspirates from 52% of the study population. *Haemophilus influenzae* was most common pathogen, followed by *Moraxella catarrhalis*, *Streptococcus pneumoniae*, *Staphylococcus aureus*, and group A streptococci. Quantitative throat cultures had positive predictive values of 41%, 53% and 75% for *H. influenzae*, *Strep. pneumoniae* and *M. catarrhalis*, respectively, while negative predictive values were 93–98%, indicating that these three pathogens do not cause sinusitis when absent from the throat.

Keywords Children, *Haemophilus influenzae*, *Moraxella catarrhalis*, sinusitis, *Streptococcus pneumoniae*, throat cultures

Original Submission: 9 May 2004; **Revised Submission:** 13 December 2004; **Accepted:** 23 December 2004

Clin Microbiol Infect 2005; **11**: 407–410
10.1111/j.1469-0691.2005.01132.x

Sinusitis is a common complication of upper respiratory tract virus infection and allergic inflammation [1]. Sinus cavity aspiration is the most

Corresponding author and reprint requests: A. Ilki, Marmara University, Faculty of Medicine, Department of Microbiology, Tibbiye Cad. No. 49, 81326 Haydarpasa, Istanbul, Turkey
E-mail: ailki@superonline.com

reliable method of determining the aetiology of sinusitis, although this is invasive and difficult to perform, particularly in children. In acute bacterial sinusitis, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, *Staphylococcus aureus* and viridans streptococci are the predominant organisms [2–4]. In chronic sinusitis, anaerobic bacteria and *S. aureus* are thought to predominate, although no or few anaerobes may be identified [5]. The present prospective study was undertaken to evaluate the microbiology of sinusitis and to determine whether a correlation exists between isolates obtained from sinuses and those from throat culture.

The study included 90 children (aged 2–9 years; 62% male) who received surgery for adenoid hypertrophy at Marmara University Hospital during a 2-year period. They were diagnosed with chronic sinusitis ($n = 21$), acute sinusitis ($n = 28$) or chronic tonsillitis and/or adenoiditis without sinusitis ($n = 41$). Informed consent was obtained from parents. Sinusitis was diagnosed by the presence of two major criteria (purulent nasal discharge, purulent pharyngeal discharge and cough) or one major plus one minor criterion (periorbital oedema, facial pain, tooth pain, earache, sore throat, wheezing/snoring, headache, foul breath, fever) [6]. The diagnosis of chronic adenoiditis was made on clinical grounds alone [7].

Throat swabs were obtained on the day of surgery and maxillary sinus aspirates were obtained during surgery. Throat swabs were suspended in 1 mL of sterile saline and diluted serially to 10^{-5} . Undiluted and diluted specimens were inoculated on to Columbia agar (bioMérieux, Marcy l'Etoile, France) and Polyvitex chocolate agar (bioMérieux) with a 0.01-mL calibrated loop and incubated in CO_2 5% v/v at 35°C for 24 h. Sinus aspirates were also inoculated both qualitatively and quantitatively on to the above media. Additionally, sinus aspirates were inoculated on to Schaedler agar (bioMérieux) and incubated at 35°C for 48 h in an anaerobic GEN box (bioMérieux) to detect anaerobes. Colonies from each plate were tested for aerotolerance. Primary plates were incubated for a further 5 days to detect slow-growing organisms. Isolates were identified by conventional methods [8]. Antibiotic susceptibility testing of *H. influenzae*, *M. catarrhalis* and *Strep. pneumoniae* was performed by disk diffusion. Penicillin susceptibility

in *Strep. pneumoniae* was tested by Etest (AB Biodisk, Solna, Sweden). Nitrocefin was used to detect β -lactamase production in *H. influenzae* and *M. catarrhalis*.

Sinus aspirates from 31 (63%) of 49 patients with sinusitis in addition to chronic tonsillitis and/or chronic adenoiditis yielded growth, whereas 16 (39%) sinus aspirates from 41 patients with chronic tonsillitis and/or adenoiditis without sinusitis were positive. The following combinations of clinical symptoms correlated significantly ($p < 0.05$) with positive sinus aspirates by Fisher's Exact Test (GraphPad InStat v. 2003; GraphPad Software, San Diego, CA, USA): coughing plus wheezing/snoring; purulent nasal discharge plus wheezing/snoring; and purulent pharyngeal discharge plus headache (Table 1).

Positive sinus aspirates ($n = 47$) yielded 72 isolates; 27 (57%) yielded a single pathogen. The predominant isolates were *H. influenzae* (40%), *M. catarrhalis* (34%), *Strep. pneumoniae* (26%) and *Staph. aureus* (17%). In three (6%) cases, anaerobic bacteria caused mixed infection. A further three (6%) cases yielded a group A streptococcus, which was the sole isolate from one patient. The predominant anaerobic isolates were *Prevotella* spp., *Porphyromonas* spp. and *Peptostreptococcus* spp. Among sinus isolates, β -lactamase was produced by 16% of *H. influenzae* isolates and 70% of *M. catarrhalis* isolates. No *Strep. pneumoniae* isolates with high-level penicillin resistance were detected, although 17% had intermediate resistance.

When throat cultures yielded *H. influenzae*, *Strep. pneumoniae* and *M. catarrhalis* at $\geq 10^2$ CFU/mL, positive sinus aspirates were recorded in 95%, 75% and 71% of cases, respectively.

Table 1. Correlation between major and minor clinical symptoms of sinusitis and sinus aspirate culture

Combination of symptoms	Sinus aspirate culture		p value
	Positive ($n = 31$)	Negative ($n = 18$)	
Coughing + PND	21	12	1.00
Coughing + PPD	16	12	0.38
PND + PPD	13	10	0.39
Coughing + headache	12	9	0.55
Coughing + snoring	26	9	0.01
PND + headache	9	7	0.53
PND + snoring	24	7	0.01
PPD + headache	5	8	0.04
PPD + snoring	18	7	0.24

PND, purulent nasal discharge; PPD, purulent pharyngeal discharge.

Table 2. Correlation between quantitative throat culture of *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis* and sinus aspirate culture

Throat colonisation ($\geq 10^2$ CFU/mL)	Sinus aspirate culture					
	<i>Haemophilus influenzae</i>		<i>Streptococcus pneumoniae</i>		<i>Moraxella catarrhalis</i>	
	Positive n (%)	Negative n (%)	Positive n (%)	Negative n (%)	Positive n (%)	Negative n (%)
Positive	18 (95)	26 (37)	9 (75)	8 (10)	12 (71)	4 (5)
Negative	1 (5)	45 (63)	3 (25)	70 (90)	5 (29)	69 (95)
Total	19	71	12	78	17	73

ively, giving positive predictive values of 41%, 53% and 75%, respectively (Table 2). Negative predictive values of throat cultures for positive sinus aspirates were 93–98%. These values were altered minimally by increasing the cut-off figure to $\geq 10^5$ CFU/mL.

The major clinical problem when considering a diagnosis of sinusitis is the differentiation of uncomplicated upper respiratory tract infection from a secondary bacterial infection of the sinuses. Transillumination, radiographic findings and sinus aspiration can confirm clinically diagnosed sinusitis. However, routine use of X-rays to diagnose uncomplicated sinusitis is not recommended for children. Upper respiratory tract infections, including sinusitis, are a leading cause of antibiotic overuse; thus, knowledge of the aetiology of sinusitis is important, since clinical symptoms may be varied, particularly in cases of chronic sinusitis [9,10]. The reference test, sinus puncture, cannot be used routinely; therefore, new strategies are needed [11,12].

In the present study, the correlation between the results of throat and sinus cultures in children was not sufficient to allow throat culture to be recommended for the bacteriological documentation of sinusitis. Similarly, Wald *et al.* [13] could not find a correlation between throat, nasopharyngeal and sinus aspirates, and Orobello *et al.* [14] demonstrated only a 45% correlation between maxillary sinus and nasopharyngeal cultures. Sener *et al.* [15] reached a similar conclusion when comparing maxillary sinus cultures with nasopharyngeal and throat cultures. The present study found that the positive predictive value of *M. catarrhalis* colonisation (75%) was higher than that of the other potential pathogens, but was not sufficient to predict the aetiology of sinusitis. In the studies mentioned above, com-

parisons were based on qualitative evaluation only, although neither qualitative nor quantitative cultures could be used for accurate prediction of the aetiology of sinusitis in the present study. Only a clinical diagnosis based on combinations of one major and one minor clinical symptom correlated with growth of sinus cultures. In contrast, negative predictive values were 93–98%, indicating that bacteria which do not colonise the throat cannot be the cause of sinusitis. Gehanno *et al.* [16] have reported that nasopharyngeal cultures have a low positive predictive value for middle ear fluid cultures, but a markedly higher negative predictive value. Patients diagnosed clinically with chronic adenoiditis without sinusitis should be evaluated more carefully for accompanying sinusitis, as 39% yielded bacterial growth from sinus aspirates. *H. influenzae*, *Strep. pneumoniae* and *M. catarrhalis* were the most common microorganisms isolated from sinus aspirates, and were susceptible to most of the antibiotics used commonly in the treatment of sinusitis [17,18].

Finally, it should be noted that the present study was performed in a university hospital and was based on sinus puncture in children. Therefore, these findings may be more applicable when sinusitis is complicated or unresponsive to treatment.

REFERENCES

1. Wald ER. Microbiology of acute and chronic sinusitis. *Immunol Allergy Clin North Am* 1994; **14**: 31–45.
2. Wald ER. Sinusitis in infants and children. *Ann Otol Rhinol Laryngol* 1992; **155**(suppl): 37–41.
3. Lund VJ. Bacterial sinusitis: aetiology and surgical management. *Pediatr Infect Dis J* 1994; **13**(suppl 1): 58–63.
4. Weinberg EA, Brodsky L, Brody A, Pizzuto M, Stiner H. Clinical classification as a guide to treatment of sinusitis in children. *Laryngoscope* 1997; **107**: 241–246.
5. Wald ER. Chronic sinusitis in children. *J Pediatr* 1995; **127**: 339–347.
6. Shapiro GG, Virant FS. Medical management in children. *Immunol Allergy Clin North Am* 1994; **14**: 47–68.
7. Kornblut AD. Non-neoplastic diseases of the tonsils and adenoids. In: Paparella MM, Shurnick DA, Gluckman JL, eds. *Otolaryngology*, 3rd edn. Philadelphia, PA: WB Saunders, 1991; 2129–2147.
8. Koneman EW, Allen SD, Janda WM, Schreckenberger PC, eds. *Color atlas and textbook of diagnostic microbiology*, 5th edn. Philadelphia: Lippincott, 1997.
9. Chan J, Hadley J. The microbiology of chronic rhinosinusitis: results of a community surveillance study. *Ear Nose Throat J* 2001; **80**: 143–145.

10. Slack CL, Dahn KA, Abzug MJ, Chan KH. Antibiotic-resistant bacteria in pediatric chronic sinusitis. *Pediatr Infect Dis J* 2001; **20**: 247–250.
11. Gwaltney JM, Scheld WM, Sande MA, Sydnor A. The microbial aetiology and antimicrobial therapy of adults with acute community-acquired sinusitis: a fifteen-year experience at the University of Virginia and review of other selected studies. *J Allergy Clin Immunol* 1992; **90**: 457–461.
12. Gwaltney JM. Acute community-acquired sinusitis. *Clin Infect Dis* 1996; **23**: 1209–1223.
13. Wald ER, Milmo GJ, Bowen A, Ledesma-Medina J, Salamon N, Bluestone CD. Acute maxillary sinusitis in children. *N Engl J Med* 1981; **304**: 749–754.
14. Orobello PW, Park RI, Belcher LJ *et al.* Microbiology of chronic sinusitis in children. *Arch Otolaryngol Head Neck Surg* 1991; **117**: 980–983.
15. Sener B, Hascelik G, Onerci M, Tunckanat F. Evaluation of the microbiology of chronic sinusitis. *J Laryngol Otol* 1996; **110**: 547–550.
16. Gehanno P, Lenoir G, Barry B, Bons J, Boucot I, Berche P. Evaluation of nasopharyngeal cultures for bacteriologic assessment of acute otitis media in children. *Pediatr Infect Dis J* 1996; **15**: 329–332.
17. Lindbaek M. Acute sinusitis: guide to selection of antibacterial therapy. *Drugs* 2004; **64**: 805–819.
18. Leung AK, Kellner JD. Acute sinusitis in children: diagnosis and management. *J Pediatr Health Care* 2004; **18**: 72–76.

RESEARCH NOTE

No evidence of *Legionella* infection in general practice patients presenting with acute respiratory infections in The Netherlands

B. M. W. Diederer¹, C. M. A. de Jong¹,
I. Aarts¹, M. F. Peeters¹, A. B. van
Gageldonk-Lafeber², B. Wilbrink² and
A. van der Zee¹

¹Laboratory of Medical Microbiology, St Elisabeth Hospital, Tilburg and ²National Institute of Public Health and the Environment, Bilthoven, The Netherlands

Corresponding author and reprint requests: B. M. W. Diederer, Laboratory of Medical Microbiology, St Elisabeth Hospital, PO Box 747, 5000 AS Tilburg, The Netherlands
E-mail: b.diederer@elisabeth.nl

ABSTRACT

The role of *Legionella* spp. in the aetiology of acute respiratory infections (ARIs) is largely unknown. In this case-control study, conducted in a general practitioner setting during 2000 and 2001, nose and throat samples from patients presenting with ARIs ($n = 230$) and controls ($n = 200$) were analysed for the presence of *Legionella* spp. by real-time PCR. *Legionella* DNA was not detected in any of the cases or controls. Thus, *Legionella* spp. do not seem to play a role in patients presenting with ARIs, nor were they present in patients who visited their general practitioner for complaints other than ARIs.

Keywords Acute respiratory infections, general practice, *Legionella* spp., real-time PCR, respiratory tract infection

Original Submission: 21 October 2004; **Revised Submission:** 28 December 2004; **Accepted:** 16 January 2005

Clin Microbiol Infect 2005; **11**: 410–412
10.1111/j.1469-0691.2005.01112.x

Legionella spp. are an important cause of community-acquired and nosocomial pneumonia. Since the first description of *Legionella pneumophila*, more than 40 species of *Legionella* have been identified, approximately half of which have been isolated from patients [1]. Infection with *Legionella* spp. can present as a severe pneumonia, with or without multisystem disease and high mortality, but can also present as a self-limiting influenza-like illness. Many individuals who seroconvert to *Legionella* are entirely asymptomatic [1,2].

Acute respiratory infections (ARIs) are a major cause of morbidity and mortality worldwide. Various infectious agents, especially viruses, have been associated with clinical syndromes ranging from mild disease, such as the common cold, to more severe conditions, such as pneumonia. However, no aetiological agent is found in a large percentage of cases [3]. Although ARIs are very common, there is only one report on the prevalence of *Legionella* spp. as a cause of community-acquired infections in general practice [4]. This may be because the diagnosis is not considered, existing diagnostic tests are insensitive, or legionellosis is distributed unevenly across the world. In The Netherlands, c. 200 cases of severe legio-